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Pain 1
Bedside neuromodulation of persistent pain and allodynia using caloric vestibular stimulation: an effectiveness trial

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Background: Caloric vestibular stimulation (CVS) is widely applied for neurological diagnosis but is also a safe, inexpensive, non-invasive neuromodulation technique. Case studies report short and sustained therapeutic effects of CVS in persistent pain (PP).
Objective: Conduct an effectiveness trial of CVS in phantom limb pain (n = 8), spinal cord injury pain (n = 12), complex regional pain syndrome (CRPS; n = 14) and non-specific PP (n = 4).
Patients and methods: Thirty-eight participants (19 males; mean age = 45.6 years) underwent 1–3 sessions of iced-water CVS. All but four also underwent a cold-arousal control (ice-pack to forehead). Subjective pain and light touch allodynia numerical rating scores (NRS) were collected pre- and post-CVS. The study was IRB-approved.
Results: MANOVA showed significant interaction of time by intervention ($F[2,32] = 3 \cdot 99$, $p < 0.05$). Univariate tests revealed pain scores differed significantly between CVS and control ($F[1,33] = 17.30$, $p < 0.01$). Pain was significantly lower 30 minutes post-CVS ($M = 3.44$, $SD = 2.62$) than post-control ($M = 4.25$, $SD = 2.79$; $t(33) = -3.77$, $p < 0.01$). Average reductions were 24.8% for CVS (1.13 NRS points, $SD = 1.67$) and 6.4% for control (0.29 points, $SD = 1.21$). The strongest CVS PP responses lasted up to one week. Importantly, CVS induced clinically significant allodynia reductions in three of nine CRPS patients with allodynia (10/10 to 2/10; 6/10 to 3/10; 2/10 to 0/10), lasting 24 hours to one month. CVS was well-tolerated (one patient had vomiting).
Conclusion: Examination is required of repeated CVS (several times/week for several weeks) to increase PP reductions from statistically to clinically significant, increase the proportion of clinically significant allodynia responders, and assess other allodynia conditions (e.g. post-herpetic, trigeminal and occipital neuralgia).

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Pain 1
Investigating a novel mechanism of hypersensitivity induced by exclusive damage to intraepidermal nerve fibres: neuropathic pain in epidermolysis bullosa

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Introduction: Small fibres that innervate the skin are especially susceptible to damage, however, their role in the development of neuropathic pain (NP) is still unclear. We are investigating pain in Epidermolysis-Bullosa-Dystrophic, (EBD) a rare disorder in which mutations of proteins of the dermo-epidermal junction lead to blistering. The somatosensory system in these patients is intact, except from the probable damage that occurs in their skin fibres.
Aims: To investigate if EBD-patients present NP and if this is due to intraepidermal fibre damage.
Methods: EBD patients were recruited within Debra Chile. The study complies with requirements of the Faculty of Medicine Ethics Committee. To detect NP we used the painDetect, DN4, NPSI questionnaires. A structured neurological examination, nerve conduction studies, autonomic tests, and Quantitative Sensory Testing (QST) was done and a skin biopsy was obtained.
Results: The prevalence of NP in EBD patients was 76.9%. Mean VAS score was 4.48 ± 0.54. QST revealed that EBD patients presented with a unique somatosensory profile with exclusive loss in thermal detection thresholds. This dysfunction presented a length dependant distribution. Nerve conduction studies (sural and motor peroneal) were normal. Testing of the autonomic system revealed no dysfunction. Quantification of IENFD of 17 EBD patients showed a significant decrease in fibre density (1.79 ± 0.8) compared with healthy volunteers (10.27 ± 0.7, $p < 0.01$).
Conclusion: These data show for the first time that EBD patients present with NP and have a small fibre neuropathy.

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Pain 1
Somatosensory evoked potentials after painful electrical stimulation in patients with central and peripheral somatosensory pathology with and without neuropathic pain

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Background: The potential clinical use of somatosensory evoked potentials after painful electrical stimulation (SSEPs) has not been systematically explored.
Objective: To compare SSEPs in controls and patients with central nervous system (CNS) lesions and Polyneuropathies (PNP) affecting the somatosensory pathway with and without neuropathic pain (NP).
Methods: Healthy adults n = 24. Patients (n = 77), mean age 55.8 ± 14.9 (20 to 83 years), grouped as with(+) and without(−) NP. CNS(+), n = 14/26, CNS(−), n = 12/26; PNP(+), n = 35/51, PNP(−), n = 16/51. 10 double pulse stimuli, 1 ms duration ISI 5 ms at 0.1Hz were applied twice (ring electrodes) in the middle finger and second toe. The electrical intensity was such to evoke pain (VAS 4 or more). Two superimposed averages were recorded at Cz-A1/A2. N1, P1 latencies and N1-P1 amplitude were measured. Patient consent were obtained.
Results: SSEPs were recorded in all controls subjects. Absent SSEPs in arms or legs(n): CNS(+) 8/14, CNS(−) 4/12, PNP(+)17/35 PNP(−) 8/16. There were no significant differences in the proportions of absent SSEPs between CNS and PNP, or their subgroups, nor between all with and without NP (Fisher's exact test). P1 latency was prolonged only in arms in both groups of CNS and PNP patients. N1-P1 amplitudes in legs were significantly smaller only compared to controls in both subgroups of CNS and PNP patients ($P < 0.0001$).
Conclusions: SSEPs may be useful in the assessment of patients with central or peripheral somatosensory pathways pathology but did not appear to distinguish between CNS and PNS pathology or between presence or absence of NP.